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Failing to meet the goals of periodontal recall programmes

What next?

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Periodontal and implant-related diseases comprise a group of chronic conditions that are driven by bacterial challenge from the retention of supragingival plaque and the formation of biofilm in the subgingival niche of the periodontal environment.

Chronic periodontitis is linked to a microbiota comprising a small group of around 15 of the 500 plus species that inhabit the oral cavity (96). The progression of periodontal disease either as a consequence of lack of treatment (6,71,84,87,140,144) or inadequate long-term management (11,104) results in ongoing attachment loss and bone resorption which, with time, will compromise patient-related outcomes such as tooth retention, aesthetics and dental function.

Dental implants are also susceptible to inflammatory diseases that are caused by the accumulation of biofilm. These conditions are categorised into those that are limited to the peri-implant soft tissues (peri-implant mucositis) and those that also affect the alveolar bone support (peri-implantitis). Peri-implant mucositis is a reversible, inflammatory lesion affecting the marginal soft tissues that surround osseointegrated dental implants but does not involve the resorption of the supporting bone. Conversely, peri-implantitis affects the supporting bone as well as the surrounding mucosa of a functioning implant (1,85 166).

The conventional treatment of periodontal and peri-implant diseases involves cause-related therapy comprising a home-based oral hygiene programme, together with professional management (scaling, root and implant surface instrumentation) which may be undertaken non-surgically or with surgical access to the affected sites. These anti-infective, professionally delivered treatments are, for the majority of patients, effective and lead onto longer-term

programmes of supportive periodontal care (SPC). Such recall programmes are an integral part of periodontal management (3) and are critical to prevent the progression of chronic diseases when they have been successfully stabilised following treatment. Even when teeth have been lost during the active stage of treatment, further tooth loss may be prevented if there is compliance with appropriate SPC (7,8,24,35,101,115). SPC is, therefore, largely founded on the chronic nature of the disease, the ability or inability of the patient to maintain plaque levels that are consistent with stability, and the willingness and ability of the clinician to deliver the appropriate management (44).

The aims of this paper are to outline the goals of SPC programmes both for chronic periodontitis and peri-implant diseases, to review the clinical- and cost-effectiveness of periodontal recall and to consider the additional treatment options available to the clinician when these goals have not been met. Within the context of this paper, the association of the recall programmes is with SPC rather than with the prevention of periodontal disease in otherwise healthy patients (147).

The goals of periodontal recall

The overarching **goals** of periodontal recall are to ensure that the periodontal or peri-implant tissues are maintained in a state of health, with the achievement of an acceptable degree of disease stability, patient comfort and oral function. There are differences of course as to how this goal might be achieved for periodontally affected teeth compared to implants. SPC for patients with periodontal disease starts at the post-treatment stage whereas for implants, the SPC commences after placement and at a point where no inflammatory change may yet

be present. With this in mind, the **aims** of recall for both scenarios are broadly the same and to:

- minimise or halt the progression of inflammatory disease;
- avert or postpone the loss of natural teeth (101) or implants, limiting the potential damaging biological (20), psychological (106) and financial consequences;
- provide the clinician with an opportunity to identify and treat pathology at an early stage in order to improve long-term prognosis and treatment outcomes (30).

In order to identify failing SPC at the patient level it is crucial that the parameters of success are first considered. At present there are no universally agreed clinical outcomes perhaps mainly because of the complex nature and progression of periodontal and peri-implant diseases along with their multifactorial aetiology. Further, the criteria for success may well differ between the clinicians' and patients' perspectives.

The patients' concerns predominantly relate to tooth loss and ongoing functionality, although the impact of SPC and indeed periodontal treatment on these parameters are infrequently reported (66). Such patient-centered outcomes may be either too complicated or long-term to be measurable in real-time clinical trials and conversely, the outcomes which concern clinicians may be difficult to relate to the contemporary concept of patient-centered care. Further, the current lack of clear definition between periodontal or peri-implant health makes the identification of success of SPC in the long-term quite challenging and we still rely predominantly on clinical and radiographic outcomes to ascertain levels of success and failure (9,18).

Periodontal tissues

Whilst unequivocally it is clear that the presence of plaque following periodontal intervention predisposes to progression of clinical attachment loss (6,7), there is a lack of empirical evidence to suggest a specific, minimum threshold plaque level that must be achieved perhaps because quantification of plaque is not an accurate surrogate marker to predict disease progression (28). Because of the complexity of the microflora associated with the aetiology of periodontal disease and the unequivocal role of the host response in determining risk and susceptibility, it is impossible and probably unhelpful to try to identify a universal minimum plaque level; the preferred option is to consider plaque control at the patient level whilst considering other risk factors such as smoking. Nevertheless, typical plaque scores of between 20-40% have been suggested as the goal for the majority of patients post-treatment and by implication throughout SPC (82).

Given the accepted influence of bacteria on periodontal and peri-implant diseases, there must be a strong focus on anti-infective measures. Although the biofilm rapidly reforms following debridement, repopulation of bacteria to baseline levels following periodontal treatment has been found to occur generally between 3-6 months (138), which assists in determining recall regimens. It follows that an increased frequency of recall should favour stability of the periodontal tissues.

Bleeding on probing has also been proposed as one of the most confident outcomes for determining appropriate intervals between periodontal SPC recall appointments given their strong negative predictive value for periodontal disease. Scores between 10-20% have been proposed as the threshold below which a significant reduction in disease progression and

subsequent attachment loss may be observed and stability and success may be more likely (74,80).

Periodontal sites with residual probing depths of <5mm and those at which a reduction in probing depth of 2mm is observed during SPC following periodontal treatment has been identified as a clinically relevant minimal threshold to enable the site to be identified as being non-risk of future progression (92) and the patient classified as a positive responder to SPC (114). The specific criteria used to define progression does vary however, with others suggesting for example, that progression of disease (and presumably failing SPC) is consistent with $\geq$ 2 teeth showing $\geq$ 3mm of interproximal attachment loss between 2 time points (92,150) or interproximal sites losing >1.5mm attachment during longitudinal monitoring (86). A critical search of the literature shows that consensus regarding the definition of success of SPC is clearly lacking (86,92,114,127,150). Clinically, of course, at the site-specific level, varied responses are inevitable depending, for example, on the initial depth of pocket and clinical attachment level, and also whether interventions are of a surgical or non-surgical nature (63). At sites where bleeding co-exists with an increase in pocket depth of over 1mm, then that site has a positive predictive value of 87% for subsequent development of bone and attachment loss (27).

In summary, therefore, the goals of successful periodontal recall regimens for teeth may be achieved with (114,150):

- Stabilisation of plaque scores at 20-40%;
- Stabilisation of bleeding scores at 10-25%;
- Probing depth reductions and maintenance between 1-2mm (at 30% of sites);

- Residual probing depths of <5mm;
- Gains in clinical attachment levels.

Peri-implant tissues

The goals of recall specific to peri-implant tissues are associated with the *prevention* of peri-mucositis and peri-implantitis with the prevalence of both conditions being 43% and 22% respectively (43). A key aim of recall is to allow an opportunity for early identification of the onset of inflammation and, therefore, in halting the progression of peri-mucositis to peri-implantitis (37). Again, whilst there is no consensus regarding the measures to assess the long-term performance of implants other than simply through survival or loss (99), there are various clinical parameters which may be used to assess the status and stability of the peri-implant tissues at recall.

The criteria for successful SPC can be aligned to those outlined for periodontal disease by looking to maintain optimum plaque control around implant sites recognising that biofilm removal will address the key aetiological factors in peri-implant disease (13). Again, absence of bleeding on probing has a high negative predictive value and is a particularly strong indicator of stability of the peri-implant tissues (81,89). Indeed, bleeding at implant sites is similarly predictive of active disease as it is around natural teeth (128).

The interpretation of probing depths can vary according to the depth of initial implant placement. The identification of a deep pocket alone with no other accompanying signs does not necessarily indicate the presence of pathology and concerns regarding potential

detrimental effects to the peri-implant mucosal seal have been shown to be unfounded provided a consistent probing force of no greater than 0.25N is applied (46).

Supracrestal implant platform position will likely have minimal pocketing at the time of placement when compared with subcrestal placement, and as such it has been suggested that longitudinal comparison of probing depths from baseline may best indicate whether there is disease progression (91). A minimum threshold of 1.0mm increase in probing depth has been suggested to classify a site with ongoing attachment loss (73). Further, any mobility of implants seen during SPC suggests a failure of osseointegration and implant removal is indicated. Mobility of implants during SPC, therefore, is of little use in early identification of peri-implantitis (62) although suppuration maybe more helpful in determining SPC management as it has been suggested as being indicative of bone loss of ≥3 implant threads (125).

The diagnosis of developing peri-implantitis can be confirmed through the identification of marginal crestal bone loss around the implant (130). Baseline radiographs should be taken at placement, and annually during SPC thereafter (40). There is a need to exercise caution regarding the appearance of bone loss adjacent to implants - coronal voids from over-aggressive countersinking, surgical trauma, and even the inevitable consequence of loading may all predispose to marginal crestal bone loss (135).

In summary, therefore, the goals of successful periodontal recall regimens for implants may be achieved with (108):

1. Probing depths no greater than 5mm;

2. Absence of bleeding on probing;

3. Absence of implant mobility;

4. Absence of pain/dysaesthesia;

5. Absence of continuous radiolucency surrounding implant;

6. Annual vertical loss of bone height no greater than 0.2mm.

The clinical effectiveness of supportive periodontal care

The literature is replete with studies of patients with chronic periodontitis who have been subjected to programmes of SPC. Our group has previously undertaken a systematic review to identify the more robust of these clinical trials and with the primary objective of identifying any differences between those managed intensively in specialist practice and those seen in the general dental services (54). The inclusion criteria for the systematic review were studies:

- of patients with chronic periodontitis;
- of SPC following surgical and non-surgical treatment in specialist and, or general care;
- with at least 12 months follow-up;
- with clinical attachment as the primary outcome measure.

We have recently updated our search but found no additional studies that fulfil these criteria. The observations from the 14 original studies, however, provide robust evidence for the potential clinical effectiveness of SPC (Table 1) and it is important for the clinician to be aware of the expectations of SPC before considering whether, over a period of time, their management at the patient or site levels can be considered a success or a failure.

The findings of the systematic review show clearly that patients who receive more frequent recall visits are likely to demonstrate less clinical attachment loss and even, in some instances, attachment gain when compared to those on irregular and less frequent programmes of care (7,38,72,75,105,110,126,162). With regular SPC provided every 3 or 4 months, attachment change is predominantly in the range – 0.85 [loss] to +0.25 [gain] and this is sustainable over periods of up to 12 years (7,38,72,75,105,110,117,118,126,133,162). Attachment loss occurs across buccal, lingual and interproximal sites and irrespective of whether the initial periodontal treatment was surgical, non-surgical or a combination of both (38,75,110,117,133,162). There is, however, limited evidence to suggest that greater loss occurs when SPC follows surgical intervention (12).

In response to the principal, focussed question of the systematic review (54) it was reported that frequent recall with a periodontist or dental hygienist in specialist care, or when provided as an intense programme in a dental school or hospital setting, generally leads to less attachment loss than when SPC is delivered by a general dentist (32,36,92). This difference tends to disappear when general dentists are provided with a clear prescription of the SPC that they are expected to provide and even when there is a slight deterioration in plaque control over 12 months (112).

Whilst these data are interesting and up to a point still provide the best available evidence for the expected outcomes of SPC, it is important to note that data are reported as group means (either at the full or part mouth level). This does not allow identification of those specific unstable and deteriorating sites which would be better markers of where SPC is failing at the site level and thus allow the clinician to re-evaluate and, or redeliver aspects of SPC

without having to resort to intuition. In contrast, in a 12-month study of 150 patients with moderate-to-advanced chronic periodontitis, SPC delivered in private practice was more effective than that provided in a dental school reducing sites with bleeding from 71% to 42%, reducing moderate (4-5mm) and deep (≥6mm) pockets from 22% and 7% to 3% and 0% respectively, and reducing the number of sites with attachment loss of 4-5mm and ≥6mm from 31% and 24% to 18% and 11% respectively (36). These population data provide valuable indication of the expectations and limitations of SPC on a patient level and also reinforce the concept that even with intensive SPC programmes, it is crucial to identify those individual sites where disease progression is likely. The data from this study (36) and others (92) suggest that for sites with probing depths ≥6mm and with full mouth bleeding prevalent at ≥30% sites then there is a clear risk for disease progression.

There is much less literature available to ascertain the effectiveness of SPC for dental implants and specifically the impact of SPC in preventing the progression of an established mucositis to peri-impalantitis. A key finding from the 2014 European Workshop of Periodontology concluded that a lack of long-term supportive care in patients with mucositis was associated with an increased risk of peri-implantitis (73). This confirmed earlier observations that the provision of regular SPC over 5 years was able to restrict considerably the progression of mucositis to peri-implantitis (Table 1) (37).

The cost effectiveness of supportive periodontal care

Having considered a number of treatment options for those patients who have sites that appear to be failing during SPC it is important to reflect on available literature that may provide some insight into the cost-effectiveness of these management strategies. In doing

so, we will then be able to provide patients not only with a prediction of the long-term clinical outcomes but also a simple explanation for the estimate of the costs of achieving those outcomes and maintaining a periodontally stable dentition (49).

Firstly, to assess the economic aspects of having SPC provided in general and specialist practice we have previously constructed a model to evaluate the cost-effectiveness of SPC using the data from the studies shown in Table 1 (54). This model was constructed from the perspective of a single patient over a 30-year period, being managed in the UK, and with the outcomes being the loss of tooth years and clinical attachment. SPC provided in specialist practice assumed 30-minute appointments with a dental hygienist and 3-monthly recall visits; SPC provided in general practice assumed 20-minute hygienist appointments with 6-monthly recall intervals and delivered both in private and public funded (State) care environments. Observations showed clearly, and perhaps not unexpectedly, that SPC achieves greater periodontal stability and higher rates of tooth retention when delivered more frequently in specialist practice when compared to provision by the general dental services. This improved long-term outcome, however, comes at a greater cost to the patient shown by incremental cost-effective ratios (ICERs) which represent the difference in costs divided by the difference in effectiveness between any intervention and the next best alternative (161). The ICER gives a measure of the cost per unit of effectiveness of implementing a more effective but more costly programme (161). For example, for SPC in specialist practice, the ICER was €290 for one extra tooth year saved and €1500 for 1mm less attachment loss over 30 years. Although this model was based on data from the UK, we later confirmed similar observations using data from across Europe, North America, Asia and Australasia (111). In summary, SPC delivered by a periodontist is more effective but comes at a greater cost.

In terms of cost-effectiveness, the value of SPC cannot be over emphasised. Pretzl and co-workers (116) used data from 98 patients who were in SPC for 10 years and found, unsurprisingly, that real costs for those who were regular attenders were greater than for infrequent attenders. Crucially, however, the maintenance of a tooth through regular SPC is considerably cheaper than extraction and replacement with dentures, implants or bridges and so for patients with chronic periodontitis SPC is the most cost-effective option. Indeed, for those patients who opt not to enrol in maintenance care, the cost of life-long SPC will equate approximately to the cost of extraction and replacement of only 3-4 teeth (50). One suggested approach, therefore, is to stress the financial benefit of SPC to patients and with the view of using this as a potential 'lever' to improve compliance (116).

There has been much less consideration of the health economics of supportive periodontal care for implants. In one 'real time' study set in a private periodontal practice in Norway, the mean supportive implant care (SIC) cost / implant with peri-implantitis was €110 over the entire period of follow-up whereas the mean SPC cost / tooth with periodontitis was €35. These figures related to a mean cost/implant/year of €10.2 and mean cost/tooth/year of €2.1 (48). So although the most cost-effective method for providing supportive implant care has yet to be established (132) the costs of long-term supportive care for implants has been estimated to be higher than for natural teeth (48).

What Next?

It is now relevant to consider the strategies for managing a patient for whom SPC is failing under two broad headings: modification of predictive factors and adjunctive treatments. In this context we define a predictive factor as one that is associated with a response or (in this

instance) a lack of response to a particular therapy (SPC) (29). This implies that there will be a differential benefit from SPC depending upon the *status* of the predictive factor. The modification of predictive factors is usually directed at the patient level whereas the use of adjunctive treatments is directed at the site level which depends on the sites at risk being identified. For the purpose of this review we will make the assumption that any site that does not respond to SPC has already been assessed carefully for retained or reformed deposits and any appropriate retreatment to remove these deposits has been undertaken.

Modification of predictive factors for supportive periodontal care

The principal patient-level factors that might affect the outcome of SPC are the frequency of the recall visits, whether the patient is seen by a periodontist or their general dental practitioner, smoking habit, diabetes and compliance with the SPC programme.

Smoking and diabetes

The impact of smoking on limiting the benefit of periodontal treatment is well established (for reviews see 60,77,134,149) with, in general terms, inferior clinical outcomes being observed in patients who continue to smoke (26,113); a finding reinforced by data from a recent meta-analysis of two longitudinal clinical trials (26). Quitting smoking as part of periodontal management has a significant effect on resolving probing depths (compared to non-quitting), an effect that is maintained over 9 months of SPC (113). Irrespective of smoking status, increased mean bleeding on probing during SPC has been shown, however, to be associated with disease severity and periodontal instability in both smokers and non-smokers, while smokers demonstrate lower mean BOP concomitantly with an increased prevalence of residual PPDs. Further, bleeding on probing of $\leq$ 20% in non-smokers and quitters may also

reflect disease stability, but more frequent SPC recall visits are still recommended for smokers in this category because of their higher proportion of residual probing depths (113,121). Clinical data may also be apparently contradictory: for example, in a retrospective cohort study of patients seen over 11 years for SPC, the prevalence of residual pockets $\geq$ 5mm increased in heavy smokers when compared to light or non-smokers yet a shorter, prospective study over just 3 years demonstrated significantly shallower probing depths in non-smokers compared to smokers yet no significant difference in clinical attachment levels (51). Perhaps of greater significance, however, was that neither probing depths nor attachment levels changed over 3 years when compared to the post-treatment baseline which reinforces the perception that whilst the clinical outcomes for smokers will likely be inferior to those for non-smokers, the *progression* of disease during SPC is unaffected by smoking status (51). Nevertheless, in the absence of data that identifies the clinical status of smokers who quit during SPC, we must pursue the strategy of regular recall appointments for smokers with advice to quit the habit based on the knowledge that quitting as part of periodontal treatment has a significant benefit on clinical outcomes (25,26,60,77,113,134,149). The association of smoking and compliance with SPC will be dealt with in a later section.

Another familiar and unequivocal risk factor for periodontal disease is poorly controlled diabetes and this appears to also be the case during SPC. The evidence, however, is limited with a single study of 92 patients who attended regularly for SPC over 5 years (34). There was a higher odds ratio for both progression of attachment loss [2.9] and tooth loss [3.1] amongst those with poor glycaemic control compared to those with good control or no diabetes (34). An interaction between smoking and diabetes showed vastly elevated odds ratios for disease

progression [6.2] and tooth loss [6.9] in this SPC cohort which again emphasises the importance of glycaemic control (and smoking cessation) in the long rather than the short term.

Frequency and duration of recall visits

As we have reported earlier in this review, those patients who undertake more frequent recall visits will be likely to demonstrate less clinical attachment loss when compared to those on irregular and less frequent programmes of care (7,38,72,75,105,108,110,126,127,162). When SPC is provided every 3 or 4 months over periods of up to 12 years, attachment change in the range – 0.85 - +0.25 is expected (7,38,72,75,105,110,117,118,126,127,133,162) which suggests that attachment loss of >1mm, over years rather than months, is indicative of failing SPC and more frequent recall appointments should be considered.

Regarding duration of SPC, for subjects on a programme for < 10 years only those probing depths $\geq$ 7mm were associated with a significantly higher risk for tooth loss in the longer term whereas in those subjects on SPC for 10 years or more, probing depths of 5mm and 6mm have been reported as being significantly associated with tooth loss. From a clinical point of view, this may indicate that there is a need for increased frequency of SPC as patients' age as recurrent disease becomes more prevalent (92).

Provider of supportive periodontal care

It is unrealistic to expect specialist periodontists to provide SPC on an indefinite basis and at some point in the care management, the patient will be referred back to their general

practitioner with a recommended strategy for long-term SPC. This is an area that has not been researched extensively although there is evidence to suggest that at least over the short term, stability of probing depths and bleeding on probing remain stable irrespective of where the SPC is provided (112). The longer term observations of Axelsson and Lindhe in their classic paper, however, suggest that frequent recall and intensive SPC with a periodontist rather than a general dentist will stabilise attachment levels and lead to less tooth loss over 6 years (7). Further, in a retrospective cohort study over 11 years, Matuliene and colleagues reported that those patients who were treated and followed through SPC in a University clinic environment received a greater frequency of recall appointments and had significantly fewer probing depths of 5mm when compared to those patients discharged to their private dentists (17% vs 30%) (92). These data don't allow an unequivocal consensus to be made with any confidence regarding the best time during an SPC programme for the specialist to transfer responsibility of care to the general dentist and it is possibly best concluded that, until further evidence from longitudinal studies becomes available, each case should be considered on an individual basis. The issue of compliance will be covered in more detail in the following section but of relevance here is the observation that the majority of 61 patients interviewed after periodontal treatment continued to attend their general dentist rather than the specialist office for SPC (47). The interview responses suggested that most patients are confident that their own dentist has the ability and skill set to maintain their periodontal health and the authors suggested that general dentists who diagnose disease and refer to a periodontist may have a particular interest in periodontology, may employ a dental hygienist and, therefore, be in a better position and confident to deliver SPC (47).

Compliance with supportive care

Supportive periodontal care

Compliance with SPC is clearly an essential prerequisite of long-term periodontal stability and maintenance of a functional dentition yet the levels of compliance are often below 50% (42,47,79,93,102,107,122,145,164). For example, study recruiting 427 patients treated for periodontal disease in a private periodontal practice identified only about 50% of the cohort entering SPC and of these, 56% became non-attenders after 20 months and 33% became 'erratic' attenders after around 18 months leaving only 10% attending regular recall after 5-6 years (41).

There is little doubt that compliance with SPC is a complex issue and one that is of multifactorial aetiology (153). Some of these factors such as the patient's age (120), which appears to have a positive association with compliance, are not easily modified although there are a number of factors that can be modified or addressed as part of SPC. A significant negative association has been identified between smoking and compliance with SPC by Delatola and co-workers (41) and this was reinforced by Ramseier's group who demonstrated that 26% of 429 smokers never returned for SPC after periodontal treatment. Further, with respect to ultimate tooth loss, smokers with an erratic pattern of compliance with SPC presented with higher rates of tooth loss (90%) than their non-smoking counterparts (79%) whilst smokers who regularly comply with SPC had only a moderately higher rate of tooth loss (45%) when compared to non-smokers who regularly comply with SPC (42%) (33). These observations stress the need to address smoking as a risk factor as early as possible in the management plan and the need for compliance with more regular recall appointments should be stressed to patients who continue to smoke.

Supportive implant care (SIC)

The evidence for the compliance with SIC is not as extensive as for SPC but nevertheless suggests a much higher rate of compliance. For example, a 3-year study of 236 patients with 540 implants showed compliance rates of 95%, 92% and 86% at 1, 2 and 3 years respectively (53) whilst a longer study of 10-years duration demonstrated a compliance rate, albeit with only 1 recall appointment/year of 93%. Cardaropoli et al. followed 74 patients through regular recall over 5 years for periodontal and, or implant support and reported a 77% compliance rate overall but when patients had implants as well as periodontal treatment, the compliance increased to 88% (22). They suggested that the additional cost and effort of the implant provision may have increased motivation to attend regular recall appointments (22).

Adjunctive treatments for supportive periodontal care

Clearly, repeated instrumentation at non-responding sites during SPC will compromise root substance and lead to an increase in tooth sensitivity. Additional treatments that do not necessitate empirical root instrumentation include the application of locally delivered antimicrobials which have now been available for over 30 years. Multiple drugs have been incorporated into these products which have been thoroughly investigated but mainly in clinical trials involving the treatment of chronic periodontitis rather than in SPC. When used in various formulations (gels, strips, chips, powders, microspheres and fibres) at the treatment stage, the mean effect sizes for improvement in clinical attachment have been reported as 0.12mm for metronidazole gel (57,78,83,97,109,124,143), 0.16mm for chlorhexidine (19,58,61,69,90,142,154), 0.24mm for tetracycline (52,55,70,78,83,90,100,151,154) and 0.46mm for minocycline (56,64,78,155,160,163). It is a reasonable assumption of course to anticipate that the effects of using such applications for

specific, failing sites during SPC will be similar to those effects reports during the definitive treatment stage and although more robust data over extended periods are still needed, there is some evidence that suggests a potential role for the reapplication of local antimicrobials during SPC. Repeated applications of microencapsulated minocycline with instrumentation at 3 and 6 months during SPC produced sustained and significantly better reductions in probing depth after 9 months than instrumentation alone (163). Similarly, greater improvements in probing depths and clinical attachment were seen after 15 months in patients with reapplications of a 2% minocycline ointment at 1 month following treatment and then at 3-monthly intervals for 12 months (156) although an earlier study of similar design had failed to show any advantage of using the antimicrobial over the same period (148).

Slow release doxycycline has also been used as an adjunctive administration during SPC (14,39,152). A significant adjunctive effect in reducing probing depths (with bleeding) >5mm and attachment levels at 3 months and a benefit for >6mm probing depths after 6 months has been reported, although these effects were not maintained at 12 months which clearly demonstrates the relatively short-term benefit in reducing inflammation and suggests that their use during SPC might have severe limitations unless reapplications are made (152). A similar observation was also made when a single application of doxycycline was used as an adjunct to instrumentation at furcation sites where improvement at 3 months could not be sustained after 6 or 12 months and the local delivery failed to reduce the need for additional instrumentation at non-responding sites after the 12 months of the study. Further, local application of doxycycline repeated annually over 3 years failed to show any advantage over instrumentation alone in maintenance patients either from a clinical or microbiological standpoint (14). These data, therefore, both for minocycline and doxycycline are promising

but by no means unequivocal to support the use of these antimicrobials in patients undergoing SPC.

Cost-effectiveness of locally delivered antimicrobials

As part of any future evaluation, consideration must be given to the health economics of using local-delivery agents as several reapplications might be indicated over a period of time. The cost-effectiveness, cost benefit and patients willing-to-pay for these treatments are paramount to their pragmatic inclusion in a programme of care. Slots and Jorgensen were amongst the first to suggest that the costs of acquiring and administering drugs can be relevant in selecting which particular drug regimen to use as an intervention and particularly if the agents demonstrate equal efficacy and toxicity (137,139). Based on this suggestion, we reported an economic analysis using a model of SPC with effectiveness data after 12 months (17,65), a previously published profile of patients' characteristics (5), a direct source of treatment costs (in the UK) and a rationale for treatment regimens involving the use of adjunctive antimicrobials (Fig. 1) (61). One comparison arm for the model was the provision of SPC without adjunctive treatments (Fig. 1). All of the real economic costs of the initial management and subsequent application of local antimicrobials during SPC are shown in Table 2 and based on UK economic data from 2009.

The calculations for the cost-effectiveness of localised antimicrobials (minocycline gel, chlorhexidine chips or metronidazole gel) during treatment and subsequently during SPC showed that the adjunctive use of metronidazole gel is less effective and more expensive than the adjunctive use of chlorhexidine chips (and therefore is unlikely to be cost-effective). The adjunctive use of minocycline is more expensive than chlorhexidine chips, but the additional

cost, per mm gain in attachment level, is less. With this particular model, minocycline was cost-effective if a patient values a mm in attachment gain on all affected teeth at a minimum cost of £1,800 in total. If a patient values this gain between £1,500 and £1,800 then SPC alone was cost-effective; for such a patient the additional gain from adjunctive minocycline did not justify the additional cost. So in summary, and although this was only a model, the principal observation was that localised adjunctive antimicrobials can deliver nearly 1 mm of attachment gain throughout SPC, but there is an additional cost. In general terms, the use of localised antimicrobials adds significantly more cost to the SPC programme. Their effects can be clinically significant but the extent to which they are used in SPC will depend predominantly on the patients' willingness-to- pay.

A systematic review and meta-analysis regarding the efficacy of adjunctive local administration of antimicrobials in the treatment of peri-mucositis has deduced that within the limitations of available studies they do not contribute any statistically significant clinical benefits (131). Several studies examine the effect of local delivery agents on peri-implantitis, although these are principally administered at the time of initial treatment rather than during the supportive care phase. The use of minocycline hydrochloride microspheres is promising (123,129) and although other agents such tetracycline solution and doxycycline powder (2), polymeric tetracycline HCl-containing fibres (95) and ornidazole (94) have been investigated, their clinical efficacy and potential role within the supportive care phase remains uncertain (68,157). Further randomised clinical trials are required whilst recognising a potential effect of the implant surface texture in compromising the efficacy of any local antimicrobial treatment (21). Furthermore, cultivated submucosal bacterial pathogens from sites of peri-implantitis have been shown to have a prevalence of resistance to common individual

therapeutic antibiotic concentrations of up to 46.7% (119). Given the current serious global concerns over the continued development of antibiotic-resistant bacterial strains (23), other adjuvant treatment strategies for SPC and SIC may then need to be explored.

The problem of the non-responding sites in patients undergoing SPC has, more recently, lead researchers to investigate the efficacy of novel mechanical, chemical and combinations of these methods in periodontal maintenance populations. For example:

- The relative effectiveness of a manual toothbrush with a fluoride dentifrice and a powered toothbrush in combination with a triclosan-containing dentifrice failed to demonstrate any clinical or microbiological superiority in 128 subjects after 1, 2 and 3 years with the patients having been scheduled for 6-monthly recall appointments (15). The introduction of a chemical plaque control intervention with 0.05% chlorhexidine and 0.05% cetylpyridium chloride was found to be effective in reducing plaque levels in a placebo-controlled trial recruiting patients who were non-compliant with plaque control (Turesky Index >1) during SPC. The study was only 3 months in duration and so the conclusions are quite limited but this observation suggests a possible option for patients who may be moved from a more intense, supportive programme to one of longer term palliative management (45).

- The use of subgingival air-polishing in recall patients has been investigated both in the short- and medium-term studies (98,165). A 2-month study failed to demonstrate differences between interventions in clinical or microbiological outcomes in 5-8mm pockets that bled on probing following either air polishing or ultrasonic instrumentation

(165); whereas a 12-month trial of 50 patients undergoing recall failed to show superiority of air-polishing with an erythitol powder in combination with 0.3% chlorhexidine although the air polishing was better accepted by the patients (98).

- Five repeated applications of photodynamic laser were delivered over a 2-week interval to a residual probing depths of ≥5mm in a small cohort of 10 maintenance patients. The variability in outcomes at different follow up time points seen in trials of other interventions is mirrored here as the laser intervention produced significant improvements in probing depths, attachment and bleeding at 6 months when compared to the control group although only the improvement in bleeding was sustained after 12 months (88). Photodynamic therapy has also been used in the early management of peri-implantitis with clinical outcomes being comparable to conventional instrumentation combined with locally-delivered antimicrobials (10,16) although their application specifically to SIC has yet to be determined.

- An Nd: YAG laser was used as an adjunct to instrumentation in probing depth sites of ≥5mm in patients on SPC for chronic periodontitis. The observations at 6-months showed no additional benefit from using the laser in this cohort (136).

Although some of the above investigations failed to show any additional benefit from using the test treatment no study showed that the test was inferior to the control intervention. When the test treatment is used instead of instrumentation (rather than as an adjunct, for example with the laser) then if such a treatment is shown to have greater patient acceptability

and is economically more cost-effective, then it may be the preferred option and especially so as it has to be reapplied on a regular basis.

A role for systemic antimicrobials?

First, it is important and pertinent to stress that when SPC or SIC appear to be failing and there are identifiable care management or behavioural factors that can be modified (frequency of recall, provider of care, smoking habit, standard of plaque control, compliance with the programme) then every attempt should be made to address these factors before making any further decisions regarding the potential role for systemic antimicrobials or consigning the patient to a programme of palliative periodontal management.

The potential role of systemic antimicrobials in the management of chronic periodontitis has been a contentious issue amongst the periodontal community and discussion and debate of the advantages and disadvantages of this role is outside the remit of this paper. Clinicians are directed to a recent pragmatic article that reviews the guidelines for the use of antibiotics in treating chronic periodontitis, the principles of dosing, selection and properties of drugs that have been used for this purpose (4). The authors identify *continuing active disease* as an indication for systemic drug therapy although fail to define this term, either clinically or microbiologically. Nevertheless, their guidance is consistent with the recommendations made by van Winkelhoff and Winkel in their excellent Commentary on the subject in which they stress that systemic antimicrobials should be used cautiously, and only after the determining the bacterial profile so that, for example, the regime of metronidazole and amoxicillin can be used for those patients with persistent infection with *Porphyromonas gingivalis* (76,159). Such reflection of course opens up the potential for using systemic

antimicrobials in those carefully selected, compliant patients on SPC or SIC for whom plaque control is exemplary and although mechanical treatment has been undertaken to the highest standard there remain multiple bleeding and or suppurating pockets in excess of 5mm. The absolutely crucial point here is the need for bacterial profiling to detect and quantify selected pathogens that are associated with disease progression prior to antibiotic susceptibility surveillance testing which, for patients on SPC, may be undertaken in the long term (every 2 years) to detect not only persistent but also changes in susceptibility profiles (158). This service is available both at academic institutions (158) and commercially (Micro-IDent®plus, Hain Diagnostics, Cookeville, Tennessee, USA) with the emphasis being on the profiling of a range of pathogens rather than just two or three known pathogens which tended to be the case with the early chair side tests.

Indeed, *Actinobacillus actinomycetemcomitans* (later *Aggregatibacter actinomycetemcomitans), Bacteroides forsythus* (later *Tannerella forsythia*) and *Porphyromonas gingivalis* (31) were identified as causative factors for periodontitis based on the 1996 World Workshop in Periodontics consensus report. Current data from the Human Oral Microbiome Database (HOMD), however, reflect the technological advances in molecular and gene sequencing techniques over the last 2 decades which have identified over 1100 bacteria taxa in the oral cavity with still 34% known only as uncultivated phylotypes (67). This increase in knowledge of the oral microbiota has served to show increased complexity and the multi-species symbiotic relationship that exists within that microbial ecosystem, which is also far more complex than previously thought. Even accepting the added complications of potential uncultivable pathogenic species, genetic variation and virulence factors, the current dogma still focuses on alteration of the bacterial ecosystem, potentially with antimicrobials

to eradicate, or maintain lower levels of periodontopathogens than needed for the progression of periodontal disease. Clinical improvement of periodontitis is associated with reductions in the levels, proportions or prevalence of associated pathogens (141) and more recent evidence has shown that the significant reductions in 30 of 40 bacterial species (including Tannerella forsythia, *Treponema denticola* and *Eubacterium nodatum*) following periodontal treatment can be maintained at 24 months (141). Thus culture and sensitivity testing across a range of bacterial species before prescribing antimicrobials certainly has potential in selected cases of failing SPC.

At the current time, there is a need for randomised clinical trials to assess the potential role of systemic antimicrobials in the supportive care of periodontitis and peri-implantitis (157). The down side of systemic antibiotics for controlling the subgingival microflora is of course their contribution to the emergence of bacterial resistance and the potential for side effects (159) and, unlike the local delivery agents, their effectiveness depends on patient compliance with dosing.

Cost-effectiveness of systemic antimicrobials

The cost-effectiveness of systemic antimicrobials as an adjunct to SPC has previously been examined (61). The analysis compared (i) no intervention at a zero cost and leading to a loss of 0.1mm CAL; (ii) SPC without systemic antimicrobials (SPC alone) costing £880 and leading to a gain of 0.5mm clinical attachment (that is an additional 0.6mm); (iii) and finally SPC with systemic metronidazole and amoxicillin (SPC+AM) costing £885 in total and giving a clinical attachment gain of 0.95mm (or 1.05mm in total over no intervention). Compared to the baseline of no intervention, SPC + AM gave an additional cost of £843 per extra mm clinical

attachment. The option of SPC without antimicrobials was only slightly less costly but significantly less effective and so not worth considering (and said to be extendedly dominated in economic terms). The data that supported our previous analysis (65) did include antimicrobial sensitivity testing but the results of this testing were not used to determine which antimicrobials should be prescribed and therefore the effectiveness results reported are therefore the same as if no antimicrobial sensitivity testing had been carried out. In addition, the costs calculated did not include any antimicrobial sensitivity testing.

For the purpose of this review, we have extended the previous cost-effectiveness analysis to include a 4th arm of SPC + antimicrobials prescribed on the basis of antimicrobial sensitivity testing (SPC+AM+ST) in addition to the three arms of no intervention, SPC alone and SPC+AM. No published data detailing effectiveness of SPC+AM+ST exist and therefore a hypothetical approach was taken to determine the additional effectiveness that adding sensitivity testing would need to yield in order to change the conclusions of a cost-effectiveness analysis. For the costs, the previous analysis took the perspective of patient in a direct payment system (i.e. all costs are passed onto the patient) and the cost of sensitivity testing was sought from two laboratories providing this service. The costs were $99 and €79.5 converted at a rate of $=£0.6955 and €=£0.7740 to give a mean cost of £65. It was assumed that the cost of different antimicrobials provided as a result of sensitivity would be negligibly different to the amoxicillin and metronidazole already costed into the SPC+AM arm. A further assumption was that even if sensitivity testing was undertaken, then all patients would still receive systemic antimicrobials prescribed 'blind' (that is, the results of sensitivity testing would influence **which** antimicrobials were prescribed rather than **whether** any should be prescribed at all).

Determining the hypothetical value at which decisions regarding the use of sensitivity testing would be influenced can be most easily explained using by plotting clinical effectiveness versus cost (Fig. 3). As the cost is known (£885+65=£950) the point of interest will lie on the interrupted vertical line. If the point lay directly on the intersection of the interrupted and solid lines the effectiveness would be 1.027mm. If the point moved along the vertical uninterrupted line so that it was **above** the solid line, (that is the effectiveness was greater than 1.027mm or 0.077mm greater than SPC+AM) then it would always be worth undertaking sensitivity testing as this approach would only be slightly more costly for significantly more effect than SPC+AM (SPC +AM is extendedly dominated in economic terms). If the point moves along the interrupted line to a point **below** the solid line but still greater than effectiveness of SPC+AM, then a judgement would need to be made as to whether the extra effectiveness was worth the extra cost. If the point moved further down the interrupted line to below the effectiveness level of SPC+AM, then SPC+AM would be more effective and less costly and so it would be inappropriate to prescribe SPC+AM+ST (SPC+AM+ST would be dominated by SPC+AM).

Conclusion

When SPC appears to be failing despite successful modification of predictive and behavioural factors then there are several factors that the clinician may consider. Increasing the frequency of recall visits and reverting to specialist management may help to stabilise the disease whilst there is sparse evidence as yet for the benefit of additional treatment methods such as laser therapy or air polishing over conventional root instrumentation. Application of local antimicrobials is an option at specific failing sites and systemic application of

antimicrobials may be considered as a clinically and cost effective regimen based, where possible, upon culture and sensitivity testing.

Abstract for Online Publication

Supportive periodontal care is a crucial aspect of the management of chronic periodontitis and peri-implantitis and is inevitably a long-commitment for both the clinician and the patient. The principal goals of supportive care are to achieve a high standard of plaque control, minimise bleeding and maintain pockets at less than 6mm. Gain of attachment around natural teeth during SPC has been reported although gain of attachment and gain of bone during supportive care may be a more pragmatic and aspirational aim in the longer term. Further, we occasionally see patients for whom, despite excellent home and professional care (surgical or non-surgical) including the management of risk factors, SPC appears to be failing and the clinician needs to consider further management options. This review considers in particular the options of using local or systemically-delivered antimicrobials to eradicate periodontal and peri-implant disease progression and discusses the extent to which culture and sensitivity testing prior to the prescription of systemically-delivered antimicrobials may be a cost-effective alternative to prescribing 'blind'.

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Tables and Figures

Table 1

<table>
<tr><th>Study/Year</th><th>No. of participants</th><th>Mean age (SD) range</th><th>Treatment</th><th>Supportive care and recall</th><th>Clinical effectiveness</th></tr>
<tr><td>Nyman et al. (1975) (105)</td><td>20</td><td>Unreported</td><td>OHI and surgical pocket elimination</td><td>Test group: Professional tooth cleaning every 2 weeks for 2 years. Control group: Scale and polish every 6 months</td><td>Test group: mean (SE) gain of CA 0.1 (0.25mm) at years. Control group: mean (SE) loss of CA – 2.2 (0.39) at years. CAL measurements relative to pre-surgical measurements</td></tr>
<tr><td>Ramfjord et al. (1975) (117)</td><td>82</td><td>39.6 (13.1) [19-61]</td><td>OHI, ScRP followed by either: curettage OR MWF OR pocket elimination with split-mouth design</td><td>Prophylaxis every 3 months for 5 years</td><td>CAL (mm) between 1 and 5 years:<table><tr><td>Curettage</td><td>Buccal</td><td>Lingual</td><td>Interproximal sites</td></tr><tr><td></td><td>-0.50</td><td>-0.65</td><td>-0.64</td></tr><tr><td>MWF</td><td>-0.26</td><td>-0.36</td><td>-0.23</td></tr><tr><td>Pocket elimination</td><td>-0.33</td><td>-0.24</td><td>-0.27</td></tr></table></td></tr>
<tr><td>Axelsson & Lindhe (1981) (6)</td><td>90</td><td>52</td><td>OHI, ScRP followed by MWF</td><td>Recall group: 30 min. appointment for Sc OHI every 2 months for 2 years and then every 3 months for 4 years. Non-recall group: referred to dentist with written instructions for plaque control programme</td><td>Baseline taken as 2 months after surgery
Mean (SD) No. teeth<table><tr><td>Recall</td><td>Baseline</td><td>19.6 (7.0)</td></tr><tr><td></td><td>6 years</td><td>19.4 (7.0)</td></tr><tr><td>Non-recall</td><td>Baseline</td><td>18.0 (5.0)</td></tr><tr><td></td><td>6 years</td><td>17.3 (5.5)</td></tr><tr><td colspan="3">Δmean CAL during 6 year period of SPC</td></tr><tr><td></td><td>Recall</td><td>+0.2</td></tr><tr><td></td><td>Non-recall</td><td>-1.8</td></tr></table></td></tr>
<tr><td>Pihlstrom et al. (1981) (110)</td><td>17</td><td>43 [22-59]</td><td>OHI, ScRP followed by MWF for half mouth</td><td>60 min. appointments with hygienist every 3-6 months for supra and subgingival scaling for 4 years</td><td>8 teeth extracted during active therapy. 6 teeth extracted during SPC. Change in mean CAL (mm) during 4 years of SPC according to initial pocket depth.<table><tr><td></td><td>ScRP</td><td>MWF</td></tr><tr><td>1-3mm</td><td>-0.22</td><td>-0.23</td></tr><tr><td>4-6mm</td><td>-0.27</td><td>-0.25</td></tr><tr><td>≥7mm</td><td>-0.47</td><td>-0.07</td></tr></table></td></tr>
<tr><td>Ramfjord et al. (1987) (118)</td><td>90</td><td>[24-68]</td><td>OHI, ScRP followed by pocket elimination, curettage, ScRP or MWF mouth, split mouth design</td><td>Recall for prophylaxis every 3 months for 5 years</td><td>ΔCAL (mm) during SPC according to initial pocket depth<table><tr><td></td><td>PE</td><td>C</td><td>ScRP</td><td>MWF</td></tr><tr><td>1-3 mm</td><td>-0.53</td><td>-0.64</td><td>-0.62</td><td>-0.64</td></tr><tr><td>4-6mm</td><td>-0.49</td><td>-0.42</td><td>-0.57</td><td>-0.43</td></tr><tr><td>≥7mm</td><td>-0.26</td><td>-0.41</td><td>-0.40</td><td>-0.53</td></tr></table></td></tr>
</table>

<table>
<tr><th>Study/Year</th><th>No. of participants</th><th>Mean age (SD) range</th><th>Treatment</th><th>Supportive care and recall</th><th>Clinical effectiveness</th></tr>
<tr><td>Kaldahl et al. (1988) (75)</td><td>82</td><td>43.5</td><td>Random allocation to treatment of quadrants by coronal scaling (cs), subgingival ScRP, SRP+MWF or osseous resection with ScRP</td><td>Recall with hygienist every 3 months for 2 years; ScRP in 3 quadrants. CS and prophylaxis provided to quadrant initially treated by CS.</td><td>Δmean CAL (mm) during SPC according to initial pocket depth (mm)
<table><tr><td></td><td>CS</td><td>ScRP</td><td>MWF</td><td>OR</td></tr><tr><td>1-4</td><td>-0.33</td><td>-0.29</td><td>-0.34</td><td>-1.26</td></tr><tr><td>5-6</td><td>-0.16</td><td>-0.02</td><td>-0.12</td><td>-0.01</td></tr><tr><td>≥7</td><td>-0.25</td><td>-0.03</td><td>-0.06</td><td>0.11</td></tr></table></td></tr>
<tr><td>Cortellini et al. (1994) (32)</td><td>23</td><td>[18-56]</td><td>OHI, ScRP followed by GTR at target sites of attachment loss ≥ 6mm.</td><td>Intesive OHI for 1 year, then:
Group A (n=15)
OHI, ScRP from hygienist every 3 months for 3 years.
Group B (n=8)
Sporadic SPC from general dentist.</td><td>Δmean CAL (mm) during SPC
Group A -0.01
Group B -2.80</td></tr>
<tr><td>Weigel et al. (1995) (162)</td><td>24</td><td>54
[40-79]</td><td>Hygiene phase and GTR</td><td>Supragingival ScRP and rinsing with 0.1% CHX every 3-6 months post-surgery.</td><td>Δmean CAL (mm) during SPC
Teeth Subjected to GTR -1.05
Sites subjected to GTR -0.94</td></tr>
<tr><td>Cugini et al. (2000) (38)</td><td>32</td><td>48 (11)
[29-71]</td><td>ScRP</td><td>Full mouth scaling and instruction in home care at 3 monthly intervals for 12 months post-therapy.</td><td>Δmean CAL (mm) -0.03 between post-treatment monitoring at 3 and 12 months.</td></tr>
<tr><td>Jenkins et al. (2000) (72)</td><td>39</td><td>[34-67]</td><td>ScRP</td><td>Allocated to one of two SPC regimes: Coronal scaling (CS) Subgingival scaling (SS) at 3 monthly intervals for 1 year.</td><td>Δmean CAL (mm) at 12 months
<table><tr><td>CS</td><td>-0.13</td><td>(0.19)</td></tr><tr><td>SS</td><td>-0.04</td><td>(0.18)</td></tr></table>Last observation carried forward for CS loser sites ≥2mm LOA that required SS</td></tr>
<tr><td>Becker et al. (2001) (12)</td><td>16</td><td>42
[30-57]</td><td>ScRP alone
ScRP & MWF
ScRP & Osseous resection</td><td>Post-surgery – weekly for 6 weeks for prophylaxis, then recall every 3 months for 5 years. Details of SPC intervention not reported.</td><td>Δmean CAL (mm) during 5 years SPC relative to initial pocket depth
<table><tr><td></td><td>ScRP</td><td>MWF</td><td>OR</td></tr><tr><td>1-3</td><td>-0.07</td><td>-0.35</td><td>-0.39</td></tr><tr><td>4-6</td><td>0.01</td><td>-0.72</td><td>-0.26</td></tr><tr><td>≥7</td><td>-0.09</td><td>-0.49</td><td>-0.97</td></tr></table></td></tr>
</table>

Study/Year	No. of participants	Mean age (SD) range	Treatment Phase	Supportive care	Clinical outcomes
Rosling et al. (2001) (126)	170	45.5 (8.4)	ScRP	OHI and subgingival instrumentation of pockets ≥5mm and BoP. Recall 3-4 visits a year for 12 years.	Average of 1.9 teeth/subject lost after 12 years of SCP. Cumulative ΔCAL (mm) over 12 years SPC according to sites: Buccal -0.85 Lingual -0.85 Approximal -0.80 Total -0.80
Rosling et al. (2001) (127)	148	44.5 (8.6)	ScRP	Recall for prophylaxis every 3-4 months for 12 years with ScRP at sites ≥5mm sites exited if CA loss ≥2mm at ≥4 teeth.	Average of 2.4 teeth/subject lost after 12 years SPC. Cumulative ΔCAL over 12 years SPC = -0.87mm.
Serino et al. (2001) (133)	64	Unreported	ScRP only or ScRP & MWF	OHI and subgingival instrumentation of pockets ≥5mm and BoP. Recall 3-4 visits a year for 13 years.	Mean (SD) tooth loss during SPC ScRP MWF 1.6 (1.7) 0.6 (1.1) Δmean CAL (mm) during 12 years SPC ScRP MWF -0.26 -0.25

Implants

Costa et al. (2012) (37)	212	49.7 (11.75) [20-65]	Implant Placement	5 SPC visits over at least 5 years	Mucositis progressed to implantitis: Group receiving SPC-18.0% of subjects. Group not receiving SPC-43.9% of subjects.

Table 2

Periodontal management	Cost £
Initial assessment	100
Non-surgical management based on 4 x 45 minute appointments with a dental hygienist	500
Prescription and placement of 5 chlorhexidine chips (PerioChips[1])	170
Prescription and placement of 3 chlorhexidine chips (PerioChips[1])	126
Prescription and placement of 1 chlorhexidine chip (PerioChips[1])	82
Cost of 4 applications of minocycline gel (Dentomycin[3])	270
Cost of 2 applications of metronidazole gel (Elyzol[2])	140
Maintenance visit with dental hygienist 1 x 20 minutes	70

[1] Dexcel Pharma Ltd, UK.

[2] Colgate-Palmolive, UK.

[3] Blackwell Supplies, UK.

SPC – Supportive periodontal care

Figure 1

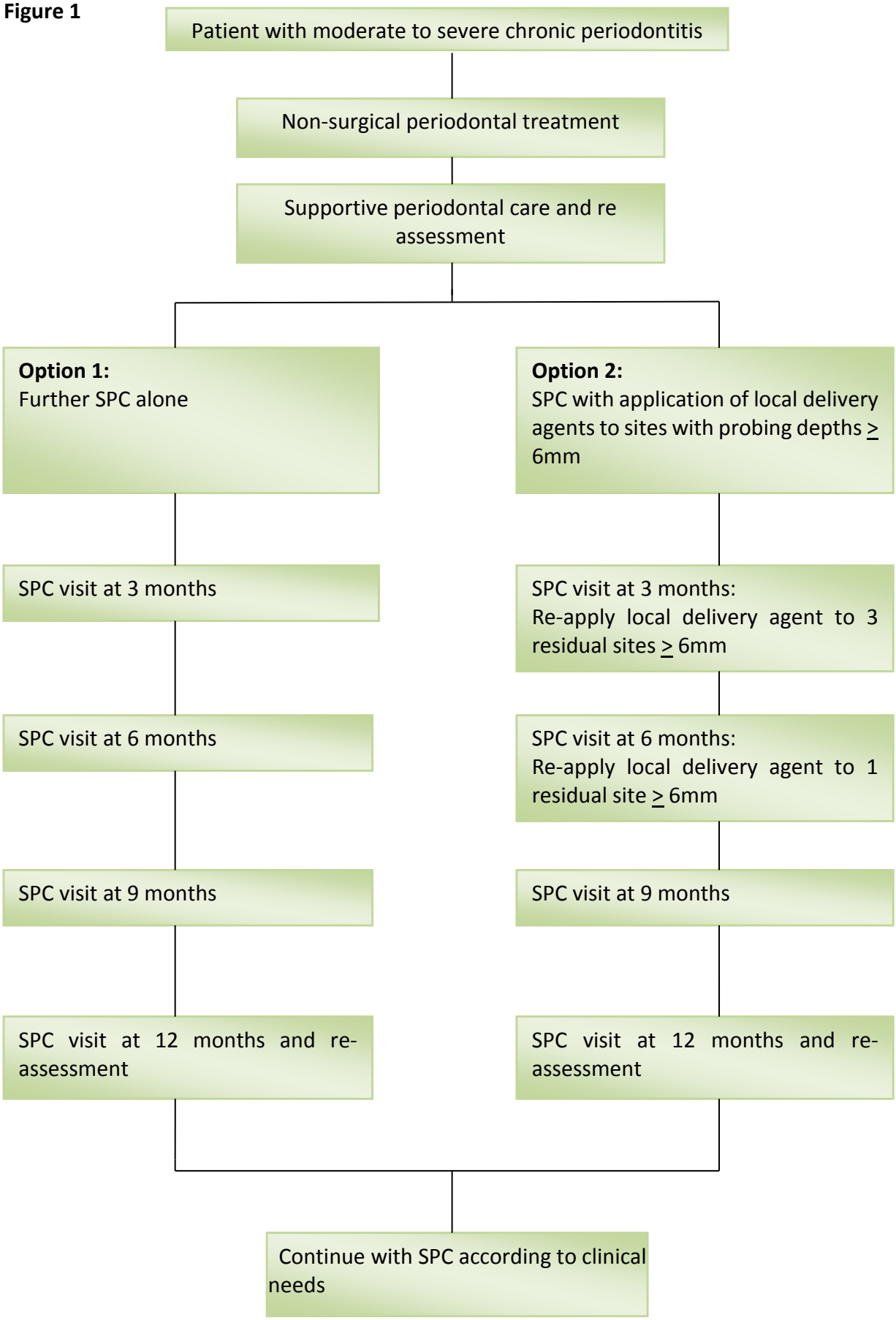

Patient with moderate to severe chronic periodontitis
Non-surgical periodontal treatment
Supportive periodontal care and re assessment
Option 1:
Further SPC alone
Option 2:
SPC with application of local delivery agents to sites with probing depths ≥ 6mm
SPC visit at 3 months
SPC visit at 3 months:
Re-apply local delivery agent to 3 residual sites ≥ 6mm
SPC visit at 6 months
SPC visit at 6 months:
Re-apply local delivery agent to 1 residual site ≥ 6mm
SPC visit at 9 months
SPC visit at 9 months
SPC visit at 12 months and re-assessment
SPC visit at 12 months and re-assessment
Continue with SPC according to clinical needs

Figure 2

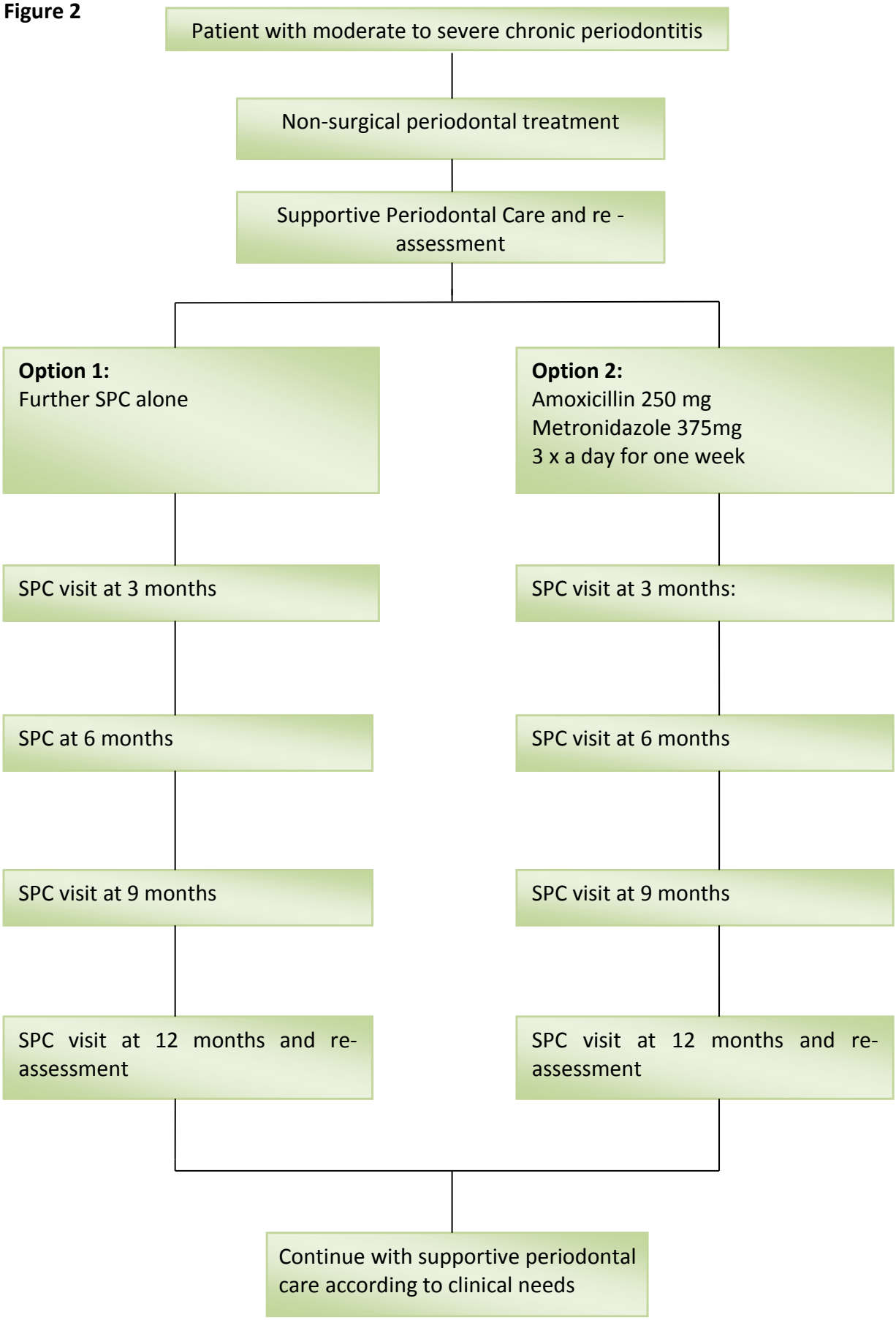
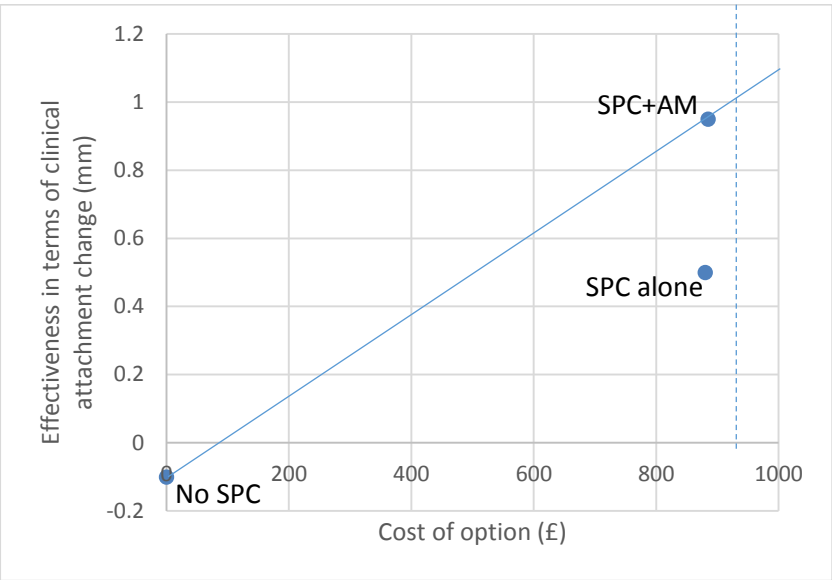

Patient with moderate to severe chronic periodontitis
Non-surgical periodontal treatment
Supportive Periodontal Care and re - assessment
Option 1:
Further SPC alone
Option 2:
Amoxicillin 250 mg
Metronidazole 375mg
3 x a day for one week
SPC visit at 3 months
SPC visit at 3 months:
SPC at 6 months
SPC visit at 6 months
SPC visit at 9 months
SPC visit at 9 months
SPC visit at 12 months and re-assessment
SPC visit at 12 months and re-assessment
Continue with supportive periodontal care according to clinical needs

Figure 3


1.2
1
0.8
0.6
0.4
0.2
0
-0.2
Effectiveness in terms of clinical attachment change (mm)
200
400
600
800
1000
Cost of option (£)
SPC+AM
SPC alone
No SPC

Legends

Table 1

Tooth loss and change in attachment during supportive periodontal care in 14 studies of periodontal disease and 1 study of implants. [Adapted from Gaunt et al.2008 (P38)].

Key: ΔCAL, change in clinical attachment level; BoP, bleeding on probing; C, curettage; CA, clinical attachment; CHX, chlorhexidine; CS, coronal scaling; GTR, guided tissue regeneration; LOA, loss of attachment; MWF, modified Widman flap; OHI, oral hygiene instruction; OR, osseous resection; PE, pocket elimination; RCT, randomized controlled trial; ScRP, scaling and root planning, standard deviation; SE, standard error; SPC, supportive periodontal care.

Table 2

Real economic costs for the management and provision of SPC by a periodontal specialist for a patient with chronic periodontitis. Management includes assessment, non surgical treatment, and prescription of 3 different locally-delivered antimicrobials during 12 months of SPC. (Data collected from 4 specialist practitioners based at different practices in the UK with costs given as British pounds 2009).

Figure 1

Suggested treatment protocol for the use of adjunctive, locally-delivered antimicrobials in the management of moderate-to-advanced chronic periodontitis. The model provides 2 Options for the use of either SPC with or without locally-delivered preparations. It is assumed that following a period of SPC there remain a number of pockets of >6mm. The locally-delivered agents are then applied to these persistent pockets of ≥6mm throughout a further period of SPC over 12 months. It is also assumed that the local agents are used according to the

manufacturers' instructions and, for example, unused gel or pastes may be reapplied and 2-weekly intervals for increased effectiveness and to avoid wastage.

Figure 2

Suggested treatment protocol for the use of adjunctive, systemic antimicrobials in the management of moderate-to-advanced chronic periodontitis. The model provides 2 Options for the use of either SPC with or without systemic antimicrobials. It is assumed that following a period of SPC there remain a number of pockets of >6mm. The systemic dose of amoxicillin 250 mg with metronidazole 375mg, 3 x a day for one week is then used only once.

Figure 3

Cost and effectiveness of different options of prescribing systemic antimicrobials (AM) as part of the SPC regimen, with and without sensitivity testing. For explanation – see text.